



Contents lists available at ScienceDirect

## International Journal of Surgery Case Reports

journal homepage: [www.elsevier.com/locate/ijscr](http://www.elsevier.com/locate/ijscr)

## Platypnea-orthodeoxia due to fat embolism

Stavros Gourgiotis<sup>a,\*</sup>, Stavros Aloizos<sup>b</sup>, Christos Gakis<sup>a</sup>, Nikolaos S. Salemis<sup>a</sup><sup>a</sup> Second Surgical Department, 401 General Army Hospital of Athens, Greece<sup>b</sup> Intensive Care Unit, 401 General Army Hospital of Athens, Greece

## ARTICLE INFO

## Article history:

Received 14 October 2010

Received in revised form 26 January 2011

Accepted 24 February 2011

Available online 8 April 2011

## Keywords:

Platypnea-orthodeoxia

Fat embolism

Dyspnea

Hypoxemia

## ABSTRACT

Platypnea-orthodeoxia is an uncommon syndrome that is characterized by dyspnea relieved by recumbency and deoxygenation following a change from a recumbent to an upright posture. We herein describe a case of platypnea-orthodeoxia in a 75-year-old man after a surgical restoration of a bitrochanteric fracture of his right femur. However, in this extremely rare case, none of the current known anatomical or functional conditions which can cause platypnea-orthodeoxia had been diagnosed. We strongly believe that this syndrome could be also the result of fat embolism.

© 2011 Surgical Associates Ltd. Elsevier Ltd. Open access under [CC BY-NC-ND license](http://creativecommons.org/licenses/by-nc-nd/3.0/).

## 1. Introduction

Platypnea-orthodeoxia is an uncommon syndrome that is characterized by dyspnea relieved by recumbency (hence the term platypnea) and deoxygenation following a change from a recumbent to an upright posture (hence the term orthodeoxia).<sup>1</sup> The diagnosis can be easily established by measuring arterial oxygen saturation in both supine and upright positions. The causes of platypnea-orthodeoxia is a right to left shunt, usually through an interatrial communication, be it an atrial septal defect or a patent foramen ovale, frequently in association with a persistent Eustachian valve. However, unless suspected, this diagnosis can be easily missed. The fact that this syndrome is often accompanied by multiple other confounding medical conditions makes its identification and management difficult. Traditionally, this condition has been reported in association with pulmonary, hepatic, and cardiac diseases. However, we report an extremely rare case of platypnea-orthodeoxia secondary to fat embolism.

## 2. Case report

A 75-year-old man presented with a bitrochanteric fracture of the right femur. He had no history of smoking, drinking, diabetes or pulmonary disease. He underwent semi-maxillectomy due to

clear cell odontogenic carcinoma, repair of inguinal hernia, and percutaneous coronary intervention for single-vessel coronary artery disease, one, two, and five year earlier respectively. Physical examination, hematologic, and blood biochemistry tests were normal. His blood pressure, heart and respiratory rate were in normal range. His heart sounds were on normal intensity with physiologic splitting and his lungs were clear to auscultation.

A surgical restoration of the fracture was performed. The second postoperative day the patient was admitted into the intensive care unit (ICU) due to hypoxemia (PO<sub>2</sub>: 55 mmHg, PCO<sub>2</sub>: 30 mmHg, SO<sub>2</sub>: 88%, breathing rate > 35 min<sup>-1</sup>) and fever (38.7 °C). Chest X-ray showed a diffuse bilateral infiltrate without pleural effusions. The patient had to be intubated and supported with invasive mechanical ventilation. A broncho-alveolar lavage (BAL) showed an increased number of macrophages containing fat; the cultures were negative. During his stay in the ICU, after his successful extubation the next day, the patient was hypoxic while sitting with a room air SO<sub>2</sub> as low as 85% (arterial pH: 7.51, PO<sub>2</sub>: 46 mmHg, PCO<sub>2</sub>: 25 mmHg). It was noticed that his hypoxia was relieved in the supine position (arterial pH: 7.52, SO<sub>2</sub>: 99%, PO<sub>2</sub>: 108 mmHg, PCO<sub>2</sub>: 35.5 mmHg). Repeat laboratory studies, which included pulmonary, adrenocortical, thyroid, liver function studies, and measurement of arterial blood gas, were all normal with no evidence of diabetic ketoacidosis, glycosuria, and dehydration.

The transesophageal echocardiogram (TEE) which was performed in both the supine and sitting position and the electrocardiogram had no pathological findings. There was normal size and function of the right and left ventricle, an estimated pulmonary artery systolic pressure of 35 mmHg, and no evidence of intracardiac shunt in the color Doppler flow imaging. There were also normal findings on a computed tomography (CT) pulmonary

\* Corresponding author at: 41 Zakinthinou Street, 15669, Papagou, Athens, Greece. Tel.: +30 2106998362; fax: +30 2106998362.

E-mail address: [drsgourgiotis@tiscali.co.uk](mailto:drsgourgiotis@tiscali.co.uk) (S. Gourgiotis).

**Table 1**  
Conditions which coexist to cause platypnea-orthodeoxia syndrome.<sup>1</sup>

1. Anatomical component
•Cardiac
Pericardial effusion
Constrictive pericarditis
•Pulmonary
Emphysema
Arteriovenous malformation
Pneumonectomy
Amiodarone toxicity
•Abdominal
Hepatic cirrhosis
Ileus
•Vascular
Aortic aneurysm
Elongation
2. Functional component
•Atrial septal defect
•Patent foramen ovale frequently in association with a persistent Eustachian valve
•Fenestrated atrial septal aneurysm

angiogram and on a high resolution CT of the chest. The CT of the chest with the administration of intravenous contrast material revealed no evidence of pulmonary embolism or pulmonary arteriovenous malformations (PAVM).

The therapeutic options included corticosteroids and continuous monitoring of SO<sub>2</sub> aiming to keep it always above 95%. There were no clinical or laboratory signs of platypnea-orthodeoxia syndrome one week later. The patient experienced an uneventful recovery and was discharged the twelfth postoperative day without symptoms. He remains stable for three years.

### 3. Discussion

Platypnea-orthodeoxia syndrome is an uncommon condition which is characterized by increased dyspnea and accentuation of arterial hypoxemia in the erect position. Dyspnea is relieved by recumbency and hypoxemia is improved by following a change from an upright to recumbent position. Cheng<sup>1</sup> reported that two conditions must coexist to cause platypnea-orthodeoxia; the anatomical condition in which there is an interatrial communication and the functional one which is the result of some factors that produce a deformity in the atrial septum resulting in a redirection of shunt flow in the upright position (Table 1).

Platypnea-orthodeoxia has also been observed in patients with liver cirrhosis or hepatopulmonary syndrome due to abnormal intrapulmonary vascular dilatation, which results in an excess perfusion for a given state of ventilation.<sup>2–4</sup> This complication is characterized by anatomical shunting and a diffusion-perfusion abnormality. In the standing position, blood flow follows gravity to the base of the lung. The abnormally dilated capillary blood vessels at the lung bases are situated far away from the alveolar epithelium, fact that decreases the oxygenation of the blood in these segments, which become more intensified in an upright position.<sup>3</sup>

Imaging methods such as angiography during CT or magnetic resonance imaging may detect abnormalities like pulmonary embolism or PAVM. Transthoracic echocardiography is highly sensitive for the presence of a ventricular septal defect while TEE is more sensitive and more likely to detect an atrial septal defect or a patent foramen ovale.<sup>5</sup> It has been reported that approximately 25% of the general population has a patent foramen ovale.<sup>6</sup>

In this reported case, none of the above anatomical or functional conditions could be demonstrated. However, there is always the possibility of a fat embolism (FE), although the BAL showed an increased number of macrophages containing fat is not high sensitive for this diagnosis. FE is defined as the occurrence of a mechanical blockage of vascular light caused by circulating droplets of fat usually getting trapped in the capillary meshwork. It mostly affects the lungs. However, the vast majority of patients subjected to FE do not present severe symptoms because the effect is only mechanical that is the simple temporary occlusion of part of the pulmonary capillary meshwork.<sup>7</sup>

Patients with FE present deep hemodynamic and respiratory changes during the most intense phase of FE such as: arterial hypertension, cardiac arrhythmia, and increased pulmonary artery pressure, pulmonary vascular resistance, and pulmonary arteriovenous shunt. All these changes may remain only for a few minutes or even days. In this reported case, no cardiac abnormality was diagnosed. However, the pulmonary artery systolic pressure was 35 mmHg, higher than normal. This acute pulmonary hypertension could probably open the close foramen as has been described by Pell.<sup>8</sup> This effect could remain only for a few moments or hours so we had no opportunity to diagnose the open foramen using TEE many hours later. On the other hand, the establishment of platypnea-orthodeoxia could be the result of the extensive lung areas in which alveoli were perfused but not ventilated due to FE ('shunt effect'). The direct result of these shunts could be the reported condition of platypnea-orthodeoxia. It may explain the fact that the patient was discharged with no symptoms and receiving no specific treatment just a week later.

### 4. Conclusions

Platypnea-orthodeoxia secondary to FE is an extremely rare occurrence. A high level of suspicion and awareness is required as the results of the diagnostic investigation are often non-specific. The establishment of platypnea-orthodeoxia could be the result of the extensive lung areas in which alveoli were perfused but not ventilated due to FE.

### Conflict of interest statement

There are no conflicts of interest.

### Funding

None.

### Ethical approval

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

### References

- Cheng TO. Platypnea-orthodeoxia syndrome: etiology, differential diagnosis, and management. *Catheter Cardiovasc Interv* 1999;**47**:64–6.
- Krowka MJ, Dickson ER, Cortese DA. Hepatopulmonary syndrome: clinical observations and lack of therapeutic response to somatostatin analogue. *Chest* 1993;**104**:515–21.
- Lange PA, Stoller JK. The hepatopulmonary syndrome. *Ann Intern Med* 1995;**122**:521–9.
- Naeije R. Hepatopulmonary syndrome and portopulmonary hypertension. *Swiss Med Wkly* 2003;**133**:163–9.

5. Faller M, Kessler R, Chaouat A, Ehrhart M, Petit H, Weitzenblum E. Platypnea-orthodeoxia syndrome related to an aortic aneurysm combined with an aneurysm of the atrial septum. *Chest* 2000;**118**:553–7.
6. Hagen PT, Scholz DG, Edwards WD. Incidence and size of patent foramen ovale during the first ten decades of life: an autopsy study of 965 normal hearts. *Mayo Clin Proc* 1984;**59**:17–20.
7. Fabian TC. Unraveling the fat embolism syndrome. *N Engl J Med* 1993;**329**:961–3.
8. Pell AC. Fat embolism syndrome (correspondence). *N Engl J Med* 1994;**330**:642–3.